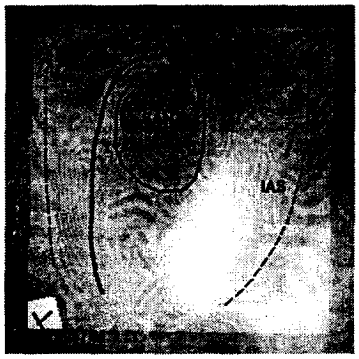

Results: Consistently, the premature stimulus shifts the slow conduction of the FPB medially toward the sinus node, increasing transverse conduction delay. This provokes rapid retrograde longitudinal conduction up the CT and preferentially enters the SAN within its superior margin. SAN depolarization then proceeds in a superior to inferior fashion.

Conclusion: Premature depolarization of the superior margins of the SAN may offer explanations for sinoatrial entrance block and the superior shift in pacemaker site after early premature stimuli. Optical mapping may allow resolution of sinoatrial conduction physiology.

977-72 Irregular Atrial Tachycardias in the Canine Sterile Pericarditis Model: Epicardial Activation Pattern

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Besides atrial flutter (AF) and atrial fibrillation (AFib), irregular atrial tachycardias (AT) demonstrating irregular "P"-waves on surface-ECG are also inducible in dogs with sterile pericarditis. Afib is due to multiple, variable, simultaneously activated wavelets, whereas a single, stable reentrant circuit is continuously activated in AF. To determine the epicardial activation pattern during irregular AT, 128 bipolar right and left atrial electrograms (electrode distance 3–8 mm) were simultaneously recorded in 6 dogs after induction of the arrhythmia. Irregular ATs were always non-sustained, with the longest episode lasting for 70 consecutive beats. Mean cycle length was 125 ± 32 s, and the mean number of consecutive beats was 36 ± 28. Circus movement reentry around pure functional or functional-anatomical obstacles was the mechanism underlying AT in all dogs. Functional obstacles were formed by long arcs of conduction block, and anatomical obstacles were provided by the orifices of the atrial vessels. As in AF, only one reentrant circuit was active during AT. However, similar to Afib, the central obstacle differed in size and location from beat-to-beat. A marked area of slow conduction was not evident. Spontaneous termination of AT was typically associated with reentry around a small central obstacle. This resulted in a relatively short revolution time of the circulating impulse, which then encountered refractory tissue.

Conclusions: Linking the single, stationary circuits in AF and the multiple, variable circuits in AFib, irregular ATs are due to single but variable reentrant circuits. The instability of this type of reentry may be contributed to the lack of a marked area of slow conduction and/or the absence of long arcs of functional conduction block.

977-73 Observations During Induced Atrial Fibrillation and its Spontaneous Termination in the Sterile Pericarditis Model

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Objectives: Atrial fibrillation (AF) is thought to be due to circulating multiple reentrant wavelets, but it is not known how interatrial activation acts to sustain AF. To test the hypothesis that the exchange of reentrant wave fronts between the right and left atria via the intercaval region, septum, and Bachmann's bundle plays a role in the self-sustaining mechanism of AF, we studied activation of both atria during induced AF and during its spontaneous termination.

Methods: Three hundred seventy-two unipolar electrograms were recorded simultaneously from an electrode array placed on both atrial free walls by using a multiplexing system during 10 induced AF episodes lasting up to 7 minutes in 5 dogs with sterile pericarditis. Activation maps were analyzed during sustained AF and just before termination of AF for 5 consecutive beats, respectively. The mean number of the lines of functional block (LFB) par beat in each atrium, the mean AF cycle length, and the mean number of wave fronts passing between the atria per beat were compared in during sustained AF and before termination of AF in the same AF episode.

Results: Complete reentrant circuits were rarely detected in either atrium during AF. Wave fronts traveling in part around LFBs were observed, and the location and length of the LFBs changed from beat-to-beat. The mean number of LFBs detected in the right atrium did not change during sustained AF compared with AF termination (1.7 ± 0.5 vs. 1.8 ± 0.8, NS). However, the mean number of LFBs in the left atrium significantly decreased from 1.4 ± 1.2 during sustained AF to 0.4 ± 0.6 before AF termination ($p < 0.05$). The mean AF cycle length significantly prolonged from 107 ± 19 ms during sustained AF to 151 ± 34 ms before AF termination ($p < 0.0001$). In the beats before AF termination, wave fronts going from left-to-right significantly decreased (1.4 ± 0.5 during AF to 0.4 ± 0.4 before AF termination, $p < 0.05$), so that the left atrium was activated by right atrial wave fronts.

Conclusions: The presence of LFBs associated with partial reentrant circuits in the left atrium and exchange of wave fronts between the atria play an important role in sustaining AF. Spontaneous termination of AF is preceded by prolongation of AF cycle length, decreasing number of LFBs in the left atrium, and a marked decrease in left-to-right activation wave fronts.

977-74 Long Electrical Silence Following Unsuccessful Internal Defibrillation Shocks of Atrial Fibrillation in Sheep

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We characterized the atrial activation patterns following unsuccessful atrial defibrillation shocks by performing epicardial mapping studies on 3 anesthetized adult sheep (heart weight 330 ± 25 gms) with atrial fibrillation (AF) induced by burst pacing. 138 silver chloride electrodes were positioned to cover the epicardium of both atria. Transvenous defibrillation coiled electrodes were positioned in the right atrial appendage (RAap), low right atrium (LRA), distal coronary sinus (CS), and left pulmonary artery (LPA) behind the left atrium. Atrial defibrillation thresholds (ADFT) were determined using 3/3 ms biphasic waveform with a step up protocol (10 volt resolution). ADFT was determined for 7 different electrode configurations: RAap→CS, RAap→LRA, LRA→LPA, RAap→LPA, CS→LRA, CS→LPA, and RAap→CS + LPA. After ADFT was determined, 15 shocks were given starting at the ADFT for each electrode configuration in each sheep. Shock strengths were decreased by 10 volts after a successful shock and increased by 10 volts after an unsuccessful shock. Pre and post-shock shock activations were recorded from all 138 electrodes using a computer assisted mapping system. For each animal 3 to 4 unsuccessful shocks just prior to or after a successful shock were analyzed for each electrode configuration. The earliest site of activation and the time from the shock to the first post-shock activation were determined. The table shows the mean ± standard deviation for the ADFT in terms of total energy (joules) and the first post-shock activation times (ms) for each electrode configuration.

RAap→CS	RAap→LRA	LRA→LPA	RAap→LPA	CS→LRA	CS→LPA	RAap→CS+LPA
0.5 ± 0.2	5.5 ± 2.4	2.0 ± 0.7	1.9 ± 1.2	2.3 ± 2.2	7.2 ± 4.0	1.2 ± 0.9
101.7 ±40.7	98.3 ±33.0	119.0 ±20.9	110.4 ±30.4	99.3 ±20.2	90.0 ±20.5	127.7 ±49.1

There was no difference ($p \leq 0.05$) in first activation times between electrode configurations. A relatively long period of electrical silence occurred following unsuccessful atrial defibrillation shocks. This appeared to be independent of electrode configuration. These findings suggest that a period of time may exist after an unsuccessful atrial defibrillation shock for further intervention.

977-75 Variations in the Termination of Atrial Flutter by Premature Beats. Studies in the Sterile Pericarditis Model

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Objectives Variations and incidence of atrial flutter (AFL) termination by single premature beats were evaluated in 26 dogs of the sterile pericarditis model.

Methods Single premature beats were introduced during stable AFL at 4 different sites in the AFL reentrant circuit, 2 at areas of slow conduction (ASC sites) and 2 at sites distant from the ASC (distant sites). The coupling interval of delivered premature beats was decremented by 2 ms from the AFL cycle length until AFL termination or local refractoriness was achieved. A multiplexing system was used to record 190 unipolar electrograms from an electrode array placed on the right atrial free wall during AFL.

Results AFL was interrupted by a single premature beat in 38/52 episodes when pacing at distant sites, and in 7/52 episodes when pacing at ASC sites ($p < 0.0001$). AFL termination was always associated with block of the activation wave front of the premature beat in an ASC. Of the 38 episodes of AFL termination by a premature beat introduced at distant sites, sinus rhythm di-